UAPM
Journal of Respiratory Diseases
and Allied Sciences

Research Article

A Study of Functional Changes of the Right Heart in Patients of COPD Acute Exacerbation with Respiratory Failure

Krupasagar Kalapala*, Mahip Saluja, Rehbar Khan

Department of Respiratory Medicine, Muzaffarnagar Medical Collge, Muzaffarnagar, Uttar Pradesh, India

Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a common and treatable disease characterized by progressive airflow limitation and tissue destruction. COPD has considerable effects on cardiac functions, primarily by affecting the pulmonary vasculature and then the right ventricle, along with the left ventricle. During acute exacerbations, hypoxemia and hypercapnia may contribute to pulmonary hypertension and right ventricular (RV) dysfunction, which adversely affect prognosis.

Methods: A prospective observational study was conducted on 61 patients admitted with acute COPD exacerbations to the respiratory intensive care unit (ICU) over a period of 6 months at a tertiary care hospital. After applying exclusion criteria, 56 patients were included. All underwent arterial blood gas (ABG) analysis, spirometry, and transthoracic echocardiography to assess RV systolic pressure (RVSP), pulmonary artery systolic pressure (PASP), right atrial size, RV ejection fraction (EF), fractional area change (FAC), and tricuspid annular plane systolic excursion (TAPSE). Clinical status was assessed with the modified Medical Research Council (mMRC) scale, COPD assessment test (CAT), BODE index, and GOLD ABCD tool.

Results: Severe COPD patients exhibited higher $PaCO_2$ (72.6 ± 5.3 *vs*. 55.2 ± 3.5 mmHg), lower PaO_2 (56.5 ± 2.96 vs. 64.2 ± 4.6 mmHg), increased right atrial area (33.3 ± 6.5 vs. 21.5 ± 5.2 cm^2), and elevated PASP (45.3 ± 3.5 vs.30.2 ± 2.3 mmHg) compared to moderate COPD. TAPSE was reduced, indicating RV dysfunction, and correlated positively with PaO_2 and negatively with $PaCO_2$.

Conclusion: COPD exacerbations were strongly associated with pulmonary hypertension and RV dysfunction. Echocardiographic evaluation of pulmonary pressures and right heart parameters provides essential prognostic information and should be integrated into COPD management.

Keywords: COPD, Acute exacerbation, Heart dysfunction, Respiratory failure.

Introduction

Chronic obstructive pulmonary disease (COPD) is recognized as a major global respiratory disorder that remains both preventable and treatable. The disease evolves primarily due to long-term inhalational exposure to noxious particles or gases, with additional contribution from host-related factors such as abnormal lung growth and development.[1] These insults result in structural alterations of the airways and alveoli, culminating in persistent airflow limitation.

Patients with COPD typically report progressive respiratory complaints, most commonly exertional dyspnoea, chronic cough, and sputum production. In addition to stable symptoms, the disease course is often complicated by acute exacerbations and the coexistence of multiple chronic systemic illnesses. The diagnosis of COPD relies on the presence of compatible symptoms and risk factors, confirmed objectively by spirometry demonstrating a post-bronchodilator FEV_1/FVC ratio below 0.7.

As COPD advances, systemic involvement becomes increasingly prominent, with a rising frequency of exacerbations and a growing burden of comorbid diseases.[2] Commonly associated conditions include cardiovascular

Access this article online

Quick Response Code

Website: uapmjournal.in

DOI: 10.70192/v3.i1.03

Address for correspondence: Krupasagar Kalapala
Department of Respiratory Medicine, Muzaffarnagar Medical Collge, Muzaffarnagar, Uttar Pradesh, India
E-mail: krupasagark@yahoo.com

How to cite this article: Kalapala K, Saluja M, Khan R. A Study of Functional Changes of the Right Heart in Patients of COPD Acute Exacerbation with Respiratory Failure. UAPM J. Respiratory Diseases Allied Sci. 2026;3(1):12-16.
Received: 18-11-2025, **Accepted:** 05-01-2026, **Published:** 12-02-2026

disorders such as ischemic heart disease, heart failure, atrial fibrillation, and systemic hypertension, along with lung cancer, recurrent respiratory infections, osteoporosis, metabolic syndrome, diabetes mellitus, gastroesophageal reflux disease (GERD), anxiety, depression, and obstructive sleep apnoea (OSA).[2-4]

Cardiac dysfunction represents a particularly important extra-pulmonary manifestation of COPD. Studies indicate that systolic or diastolic heart failure is present in approximately 20 to 70% of affected individuals, with an annual incidence estimated at 3 to 4%. Furthermore, evidence of left ventricular dysfunction has been identified in nearly 40% of COPD patients who required mechanical ventilation for type-2 respiratory failure.[1]

Chronic pulmonary pathology in COPD exerts significant effects on the right side of the heart. Persistent airway and parenchymal disease lead to chronic hypoxaemia, triggering hypoxic pulmonary vasoconstriction. Over time, this results in right ventricular dilatation and hypertrophy, eventually progressing to right ventricular failure, commonly referred to as cor pulmonale.

Pulmonary arterial hypertension (PAH) represents a progressive haemodynamic complication that may arise in this setting. It is defined by a resting pulmonary arterial pressure exceeding 25 mmHg, with pulmonary artery wedge pressure (PAWP) or left ventricular end-diastolic pressure (LVEDP) remaining below 15 mmHg and pulmonary vascular resistance (PVR) greater than 3 Wood units. In COPD, elevated PVR is largely attributed to sustained hypoxia-induced vasoconstriction, along with vascular remodelling, arterial wall thickening, and *in-situ* thrombosis. These mechanisms contribute to a progressive rise in pulmonary artery pressure, increasing right ventricular afterload and driving maladaptive right heart remodelling, ultimately culminating in right heart failure.[5]

Methods

The present study was designed to explore the relationship between respiratory failure and alterations in pulmonary haemodynamics among patients with COPD, with particular emphasis on structural and functional involvement of the right heart.

A total of 56 patients admitted with acute exacerbation of COPD to the respiratory intensive care unit of a tertiary care centre were consecutively enrolled. During hospitalisation, all participants underwent a comprehensive transthoracic echocardiographic evaluation focusing on right heart morphology, systolic performance, and pulmonary vascular pressures. Right ventricular systolic pressure (RVSP) was calculated using the standard Doppler-derived equation: $RVSP = 4(VTR)^2 + RAP$,
where VTR represents the peak tricuspid regurgitation velocity (m/s) and RAP denotes mean right atrial pressure (mmHg).

Pulmonary arterial pressure (PAP) was estimated as the product of cardiac output and pulmonary vascular resistance, with pulmonary vascular resistance (PVR) comprising arterial, capillary, and venous components. Tricuspid annular plane systolic excursion (TAPSE) was also measured to assess right ventricular adaptability and systolic reserve in COPD patients.

In addition to echocardiography, all patients underwent detailed clinical and laboratory assessment during admission. This included serial blood pressure measurements, body mass index (BMI) calculation, routine biochemical investigations such as complete blood count (CBC), liver function tests (LFT), kidney function tests (KFT), and lipid profile, along with electrocardiography (ECG), spirometry, and arterial blood gas (ABG) analysis. Functional status and symptom burden were evaluated using validated clinical tools, including the modified Medical Research Council (mMRC) dyspnoea scale, the COPD assessment test (CAT)—an eight-item instrument assessing health status impairment and the BODE index, which integrates body mass index, airflow obstruction, dyspnoea severity, and exercise capacity to reflect disease severity.

Right ventricular systolic dysfunction was defined using two-dimensional echocardiographic parameters, namely right ventricular fractional area change (RV FAC) <35% and right ventricular ejection fraction (RV EF) <44%. Echocardiographic evaluations were performed as part of the diagnostic work-up, and patients did not receive targeted therapy for pulmonary hypertension. Management was limited to standard COPD-specific treatment, including β-agonists, corticosteroids, antimicrobial therapy where indicated, supplemental oxygen, and non-invasive ventilation when clinically required.

A detailed physical examination was conducted for all patients, and comorbid conditions were identified based on clinical history, examination findings, and ongoing pharmacotherapy. All investigations were completed during the index hospital admission. Patients with significant confounding cardiovascular conditions such as severe cardiomyopathy, chronic ischemic heart disease, resistant systemic hypertension, atrial septal defect, valvular heart disease, thyroid disorders, severe arrhythmias, major electrolyte disturbances, malignancy, severe systemic illness, or prior established cardiovascular disease were excluded from the study.

Furthermore, individuals with alternative causes of pulmonary hypertension were excluded to avoid confounding, including those with interstitial lung disease, sleep-disordered breathing, alveolar hypoventilation syndromes, mixed restrictive-obstructive pulmonary disorders, chronic high-altitude exposure, vasculitis, pneumoconiosis, congenital heart disease, and pulmonary venous occlusive disease etc.

Written informed consent was obtained from all participants prior to enrolment. Continuous variables were

expressed as mean ± standard deviation and statistical comparisons were performed using Student's *t*-test.

Results

During the study period, a total of 61 patients were consecutively admitted to the respiratory intensive care unit with an acute exacerbation of COPD. Of these, six individuals were excluded due to the presence of significant complicating conditions, resulting in a final study cohort of 56 patients with confirmed COPD exacerbation.

All continuous data were expressed as mean ± standard deviation (SD). Statistical comparison between variables was performed using the paired student's *t*-test.

The study population demonstrated a clear male predominance. Comparative analysis revealed no statistically significant differences between patients with moderate and severe COPD with respect to body mass index (BMI), blood pressure levels, blood glucose values, prevalence of diabetes mellitus, or patterns of tobacco use (Table 1).

Arterial blood gas analysis demonstrated better oxygenation among patients with moderate COPD, reflected by higher mean PaO_2 values (64.2 mmHg ± 4.6) when compared with those observed in the severe COPD group (56.5 mmHg ± 2.96). In contrast, carbon dioxide retention was more pronounced in severe COPD, with significantly elevated $PaCO_2$ levels (72.6 mmHg ± 5.3) relative to patients with moderate disease (55.2 mmHg ± 3.5). Although a declining pattern of left ventricular ejection fraction (EF%) was noted in patients with severe COPD, this difference did not reach statistical significance (Table 2).

Echocardiographic evaluation of right heart dimensions was successfully completed in all 56 participants. Patients with severe COPD exhibited markedly higher pulmonary artery systolic pressure (PASP) and greater right atrial enlargement compared to those with moderate disease severity (45.3 mmHg ± 3.5 *vs* 30.2 mmHg ± 2.3; and 33.3 cm^2 ± 6.5 *vs* 21.5 cm^2 ± 5.2, respectively).

Table 1: Patient's demographics and clinical characteristics

Variables	*Total (n=56)*	*Moderate COPD (n=21)*	*Severe COPD (n=35)*	*p-value*
Male (n, %)	38 (67.8)	16 (76)	22 (62.8)	0.06
Age (years)	79.1 ± 5.1	79.2 ± 5.4	78.2 ± 4.9	0.611
Systolic BP (mmHg)	127.6 ± 16.9	125.8 ± 17.7	135.2 ± 16.2	0.156
Diastolic BP (mmHg)	72.4 ± 8.2	70.6 ± 9.8	80.2 ± 6.7	0.097
BMI (Kg/m^2)	26.2 ± 2.6	26.1 ± 3.3	26.5 ± 2.1	0.856
Current smokers (n, %)	49 (87.5)	15 (71.4)	34 (97)	0.654

(Data is expressed as mean ± standard deviation)

Table 2: The clinical and instrumental parameters of patients

Variables (mean value)	*Moderate COPD (n=21)*	*Severe COPD (n=35)*	*p-value*
FEV1%	66 ± 4.3	44.1 ± 5.2	0.062
pH	7.38 ± 0.02	7.32 ± 0.03	0.2
$PaCO_2$ (mmHg)	55.2 ± 3.5	72.6 ± 5.3	0.05
PaO_2 (mmHg)	64.2 ± 4.6	56.5 ± 2.96	0.05
HCO_3- (mmol/l)	30.2 ± 3.2	37.9 ± 5.3	0.256
SO_2%	92.5 ± 2.2	89.2 ± 2.7	0.33
EF%	51.2 ± 2.7	36.3 ± 6.3	0.652
Left atrial area (cm^2)	21.7 ± 4	27.3 ± 5	0.062
RVSP (mmHg)	38.2 ± 2.9	42.4 ± 4.7	0.06
PASP (mmHg)	30.2 ± 2.3	45.3 ± 3.5	0.05
TAPSE (mm/s)	14 ± 6	11.2 ± 2.4	0.05
Right atrial area (cm^2)	21.5 ± 5.2	33.3 ± 6.5	0.05
PVR wood units	1.6 ± 0.4	4.9 ± 1.6	0.07
CAT score	26 ± 6.4	31 ± 7.2	0.06

(Data is expressed as mean ± standard deviation)

To further examine the interaction between gas exchange abnormalities and right heart function, multivariable linear correlation analysis was performed. A significant inverse relationship was identified between arterial oxygen tension (PaO_2) and right atrial area, indicating greater atrial enlargement with worsening hypoxaemia (Figure 1). Similarly, PASP values were significantly elevated in patients with severe COPD, and a statistically significant correlation was observed between PASP and PaO_2 across the entire study cohort (Figure 2).

Assessment of tricuspid annular plane systolic excursion (TAPSE) revealed evidence of impaired right ventricular systolic performance across the entire study population, with more pronounced dysfunction observed in patients with severe COPD. Mean TAPSE values were lower in the severe disease group (11.2 mm/s ± 2.4) compared to those with moderate COPD (14 mm/s ± 6).

To explore the relationship between right ventricular performance and gas exchange, TAPSE measurements were analysed in relation to arterial blood gas parameters. This analysis demonstrated statistically significant associations between TAPSE and arterial oxygen tension (PaO_2) (Figure 3), as well as between TAPSE and arterial carbon dioxide levels ($PaCO_2$) (Figure 4).

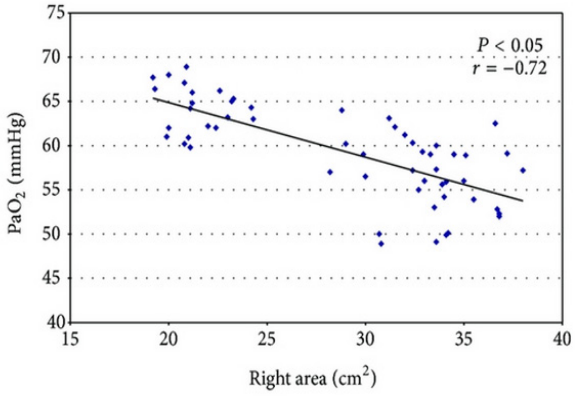

Figure 1: Correlation between values of PaO_2 and area of the right atrium.

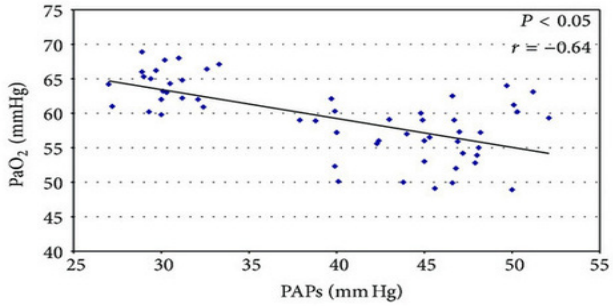

Figure 2: Inverse correlation between PaO_2 and PASP

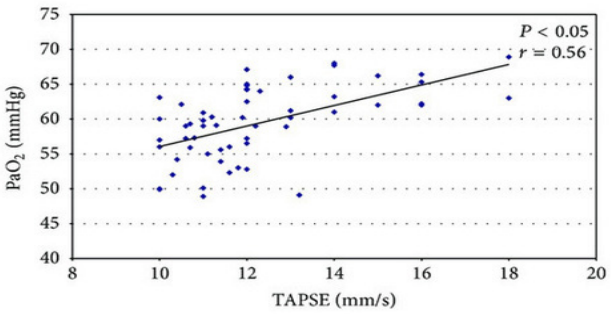

Figure 3: Direct correlation between TAPSE and PaO_2

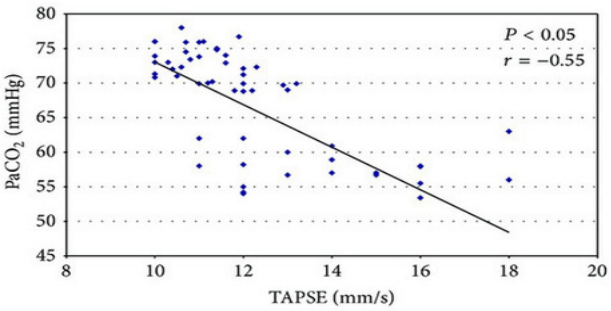

Figure 4: Inverse correlation between TAPSE and $PaCO_2$

Discussion

Progressive lung damage in chronic obstructive pulmonary disease leads to a gradual decline in effective alveolar ventilation. As ventilation worsens, sustained hypoxaemia develops, triggering hypoxic pulmonary vasoconstriction. This adaptive response, when persistent, increases pulmonary vascular resistance and ultimately contributes to the development of pulmonary hypertension.

Terzano, Romani *et al.*[6] investigated these mechanisms in a cohort of 75 patients admitted with acute COPD exacerbations. Their study incorporated transthoracic echocardiographic evaluation of right heart chambers alongside comprehensive clinical assessment, including laboratory investigations, blood pressure measurements, BMI, electrocardiography, pulmonary function testing, and health-status evaluation using the Saint George's respiratory questionnaire (SGRQ). In contrast, our study focused on symptom burden using the CAT score, BODE index, and mMRC grading among patients with moderate and severe COPD. Despite this detailed symptom evaluation, no statistically significant association was observed between the degree of airflow obstruction and symptom severity at rest. However, a significant relationship emerged when pulmonary haemodynamics were examined. Our analysis demonstrated a clear association between pulmonary arterial pressure and arterial blood gas parameters, highlighting the central role of hypoxaemia in the development of pulmonary hypertension in COPD. Additionally, the observed correlation between

right atrial enlargement and PaO_2 reinforces the concept that chronic hypoxaemia serves as an adverse prognostic marker for progression toward right heart failure. These findings underscore the importance of pulmonary hypertension and right atrial dilatation as key but often under-recognised components of COPD, since their clinical manifestations may be obscured by dominant respiratory symptoms. Consequently, Doppler echocardiography for pulmonary artery pressure estimation and right chamber size assessment should be regarded as indispensable tools in prognostic evaluation of COPD patients.[6]

The functional relevance of right ventricular assessment is further supported in a review by Bleeker *et al.*[7], that summarized multiple studies demonstrating a strong correlation between TAPSE and right ventricular ejection fraction measured by radionuclide angiography. TAPSE has been shown to be highly reproducible and to serve as a powerful prognostic indicator in heart failure.[8-10] Right ventricular ejection fraction reflects global RV performance, and in the present study, RV systolic function was assessed through TAPSE measurement, representing systolic displacement of the tricuspid annulus toward the cardiac apex.

In our cohort, right atrial dimensions were correlated with arterial blood gas values, revealing that worsening hypoxaemia was associated with smaller atrial dimensions, while elevated $PaCO_2$ demonstrated an inverse relationship with atrial size. Importantly, these associations were independent of spirometric disease severity, as defined by FEV_1, and were also unrelated to subjective dyspnoea scores. Such findings provide further evidence that the interplay between respiratory failure and right heart dysfunction extends beyond conventional markers of COPD severity.

Right ventricular impairment in COPD primarily arises from increased afterload imposed by pulmonary vascular disease. Elevated pulmonary circulation pressures not only disrupt right ventricular mechanics but also exacerbate abnormalities in gas exchange, creating a detrimental feedback loop that accelerates disease progression.[11] Our results clearly illustrate the interdependence between deteriorating ventricular function and hypoxaemia.

In summary, arterial blood gas parameters in chronic respiratory failure should be regarded not only as indicators of ventilatory impairment but also as negative prognostic markers for right heart involvement. Respiratory failure in COPD is closely intertwined with pulmonary hypertension and with both structural and functional remodelling of the right heart, reinforcing the need for integrated cardiopulmonary assessment in these patients.

References

1. Global Initiative for Chronic Obstructive Lung Disease, GOLD Guidelines, 2021. goldcopd.org.
2. Terzano C, Romani S, Paone G, Conti V, Oriolo F. COPD and thyroid dysfunctions. Lung. 2014;192(1):103-09.
3. Jenkins CR, Jones PW, Calverley PM, *et al*. Efficacy of salmeterol/fluticasone propionate by GOLD stage of chronic obstructive pulmonary disease: analysis from the randomised, placebo-controlled TORCH study. Respir Res. 2009;10(1):59.
4. Holguin F, Folch E, Redd SC, Mannino DM. Comorbidity and mortality in COPD-related hospitalizations in the United States, 1979 to 2001. Chest. 2005;128(4):2005-11.
5. Galiè N, Hoeper MM, Humbert M, *et al*. Guidelines for the diagnosis and treatment of pulmonary hypertension: the Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). Eur Heart J. 2009;30(20):2493-2537.
6. Terzano C, Romani S, Gaudio C, Pelliccia F, Serao M, Vitarelli A. Right heart functional changes in the acute, hypercapnic exacerbations of COPD. Biomed Res Int. 2014; 2014: 596051.
7. Bleeker GB, Steendijk P, Holman ER, *et al*. Assessing right ventricular function: the role of echocardiography and complementary technologies. Heart. 2006; 92 (Suppl 1): i19-i26.
8. Kaul S, Tei C, Hopkins J M. *et al* Assessment of right ventricular function using two-dimensional echocardiography. Am Heart J 1984; 107: 526–31.
9. Ghio S, Recusani F, Klersy C. *et al* Prognostic usefulness of the tricuspid annular sustolic excursion in patients with congestive heart failure secondary to idiopathic or ischemic dilated cardiomyopathy. Am J Cardiol 20008; 5: 837-42.
10. Smith JL, Bolson EL, Wong SP, Hubka M, Sheehan FH. Three-dimensional assessment of two-dimensional technique for evaluation of right ventricular function by tricuspid annulus motion. Int J Cardiovasc Imaging. 2003; 19(3): 189-97.
11. Kermanian R, Dosanjh H, Lewis MI, Matusov Y. Pathophysiology and management of right ventricular failure in critically ill patients: A narrative review. World J Crit Care Med. 2025; 14(4): 111434.